UNIVERSITY OF MINNESOTA
RECEIVED
MAY
13
1957

Journal Lancet
Lancet Publications, Inc.
Northwestern Pediatric Society
814 South 10th Street
Minneapolis 3, Minnesota

VOLUME XII January 1956 NUMBER 1

Clinical Proceedings

OF THE

CHILDREN'S HOSPITAL

WASHINGTON, D. C.

Returned to Library

TUBERCULOUS PERICARDITIS TREATED MEDICALLY AND SURGICALLY. *Sol Katz, M.D., George Rivara*..... 1

BORIC ACID POISONING. *Aaron Searle, M.D.*..... 13

ORAL INJURY BY BALL POINT PEN. *Lee M. Sackett, D. D. S., John J. Sullivan, D. D. S.*..... 16

to help you combat iron deficiency in infants...

How Fer-In-Sol improved infants' blood pictures

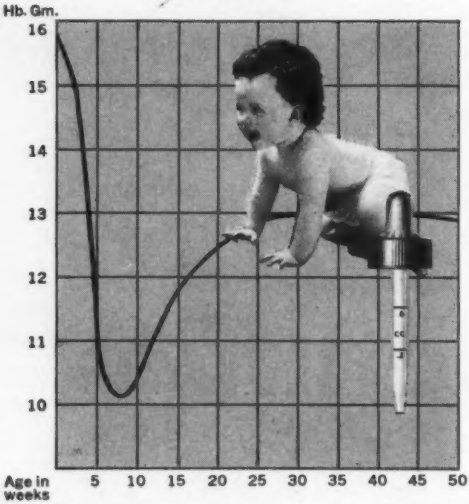

Chart adapted from Niccum, Jackson and Stearns: A.M.A. Am. J. Dis. Child. 86: 553, 1954.

Mean hemoglobin determinations of infants on a predominantly milk diet receiving Fer-In-Sol. The Fer-In-Sol dose was 5 mg. of iron (0.2 cc.) per day at 3 months, increased to 10 mg. of iron (0.4 cc.) after 6 months of age.[1]

Fer-In-Sol

iron in a drop for infants and children

"Iron deficiency anemia has a peak incidence between six months and two and one-half years..."[2]

Smith and Rosello[3] found that the peak incidence of iron deficiency in infants and children occurred between the ages of 6 and 24 months, when growth is most rapid. Sturgeon[4] reports that published data indicate iron deficiency in infants is common. Although consuming normal diets, the majority of infants in the last half of the first year of life, and throughout the second year of life, "exist in relatively severe states of iron deficiency (30% or more depleted)."[4]

Niccum, Jackson and Stearns[1] found that prophylactic administration of Fer-In-Sol in small daily doses "was sufficient to maintain hemoglobin values at a constant level throughout the latter half of infancy in all full term infants"... resulted in significantly higher hemoglobin values than did ferric ammonium citrate ... did not produce gastrointestinal disturbances.

FER-IN-SOL

Fer-In-Sol® provides a pleasant way to supply ferrous sulfate—a well tolerated, well absorbed and efficiently utilized form of iron. The vehicle is acidulous to improve absorption and utilization. The good taste of the solution makes it readily acceptable to young children. And tolerance is good.

Only 0.3 cc. of Fer-In-Sol supplies 7.5 mg. of iron—more than the Recommended Daily Allowance for children up to 4 years old. Fer-In-Sol is supplied in 15 cc. and economical 50 cc. bottles, with the new calibrated 'Safti-Dropper' of unbreakable plastic.

(1) Niccum, W. L.; Jackson, R. L., and Stearns, G.: A.M.A. Am. J. Dis. Child. 86: 553, 1954. (2) Smith, C. H.: Bull. New York Acad. Med. 30: 155, 1954. (3) Smith, N. J., and Rosello, S.: J. Clin. Nutrition 1: 275, 1953. (4) Sturgeon, P.: Pediatrics 13: 107, 1954.

MEAD | SYMBOL OF SERVICE IN MEDICINE

MEAD JOHNSON & COMPANY, EVANSVILLE 21, INDIANA, U.S.A.

MEAD JOHNSON & COMPANY, Suite 419 Eig Building, 8641 Colesville Road, Silver Spring, Maryland. JUniper 9-1222

CLINICAL PROCEEDINGS
OF THE CHILDREN'S HOSPITAL
13th and W Streets, Washington 9, D. C.

EDITORS FOR JANUARY

MARSHALL M. PARKS, M.D. J. WILLIAM OBERMAN, M.D.

EDITOR-IN-CHIEF

ROBERT H. PARROTT, M.D.

MANAGING EDITORS

FREDERIC G. BURKE, M.D.
JOSEPH M. LOPRESTI, M.D.
E. CLARENCE RICE, M.D.
SYDNEY ROSS, M.D.

ASSOCIATE EDITORS

SIMONE ALIKPALA, M.D.
JOHN BAYLY, M.D.
ROGER BERGSTROM, M.D.
STANLEY L. BLUMENTHAL, M.D.
GEORGE J. COHEN, M.D.
ALLEN B. COLEMAN, M.D.
GORDON W. DAISLEY, JR., M.D.
GRACE H. GUIN, M.D.
MARGARET GUTELUIS, M.D.
HANS G. KEITEL, M.D.
JOHN O. NESTOR, M.D.
J. WILLIAM OBERMAN, M.D.
MARSHALL M. PARKS, M.D.
ROLAND W. PENICK, M.D.
GEORGE WILLIAM WARE, M.D.
CHARLES R. WEBB, M.D.
RALPH D. WHITLEY, M.D.

GENERAL MANAGER

THELMA WALLER

CONTRIBUTING EDITORS FROM THE RESIDENT STAFF: WALTER E. AHRENS, M.D.; MICHAEL J. ARIA, M.D.; YOWALAKSN ARTHACHINTA, M.D.; CARLOS S. BERROCAL, M.D.; CONRADO BOGAERT, M.D.; JAMES A. DAVIS, JR., M.D.; ENID F. GILBERT, M.D.; JUAN F. JIMENEZ, M.D.; RENE JUTRAS, M.D.; JESUS LOPEZLIRA, M.D.; KENSUKE NISHIMURA, M.D.; NILUFER ONUR, M.D.; NATIVIDAD PANGAN, M.D.; JOSE R. PUIG, M.D.; ROBERT REID, M.D.; LUCAS RUBIO, M.D.; CARMEN SANTOS, M.D.; AARON SEARLE, M.D.; JOHN J. SULLIVAN, D.D.S.

PUBLICATIONS COMMITTEE OF THE MEDICAL STAFF: E. CLARENCE RICE, M.D.; FREDERIC G. BURKE, M.D.; HANS G. KEITEL, M.D.; PRESTON A. MCLENDON, M.D.; MARSHALL PARKS, M.D.; ROBERT H. PARROTT, M.D.; AND JOHN A. WASHINGTON, M.D.

PUBLISHED MONTHLY BY THE STAFF AND RESEARCH FOUNDATION OF THE CHILDREN'S HOSPITAL, WASHINGTON, D. C.

Cases are selected from the weekly conferences held each Sunday morning at 11:00 A.M., from the Clinicopathological conferences and from the monthly Staff meeting.

This bulletin is printed for the benefit of the present and former members of the Attending and Resident Staffs, and the clinical clerks of Georgetown and George Washington Universities.

Subscription rate is $2.00 per year. Those interested make checks payable to "Clinical Proceedings Dept.," The Children's Hospital, Washington, D. C. Please notify on change of address.

Copyright 1956, Children's Hospital

Entered as second class matter November 21, 1946 at the post office at Washington, D. C., under the Act of March 3, 1879. Acceptance for mailing at special rate of postage provided for in Section 538, Act of February 28, 1925, authorized January 17, 1947.

TUBERCULOUS PERICARDITIS TREATED MEDICALLY AND SURGICALLY

Sol Katz, M.D.
George Rivara*

Pericarditis with effusion due to *Mycobacterium tuberculosis* is being observed more often since more effective anti-tuberculous drugs are available than previously. The following case and discussion provide a summary of the diagnosis, pathogenesis and treatment. This case required antituberculous, adrenal steroid and surgical management.

CASE REPORT

T. S., a 5 year old colored boy, was admitted to the Children's Hospital August 20, 1955 with presenting complaints of dyspnea and abdominal distension for one day. He was perfectly well until three days prior to admission when a mild cough developed. One day prior to admission he was somewhat lethargic and anoretic. On the day of admission he played actively throughout the day, but gradually developed marked dyspnea and orthopnea, relieved by standing in front of an open window. His abdomen was also quite distended at this time although his mother had previously noticed that it had been increasingly difficult for him to wear his trousers.

Past history was non-contributory except for the occurrence of two nosebleeds three months prior to admission. There was no known contact with a person with tuberculosis.

On admission he appeared acutely ill and in moderately severe respiratory distress. Rectal temperature was 101°; pulse 140 per minute; respirations 60 per minute and regular; and blood pressure 124/78. He weighed 44 pounds, an increase of 11 pounds over his usual weight. Even in the erect position the neck veins were noted to be distended and pulsating. The lung fields were clear to percussion and auscultation. The heart was quite enlarged, extending from two fingers' breadth to the right of the sternum to the left anterior axillary line. The PMI was located in the fifth and sixth left interspaces in the midclavicular line. The heart sounds on admission were normal and there were no murmurs present. The abdomen was markedly distended and appeared to contain fluid. The liver was enlarged to three fingers' breadth below the costal margin, had a firm edge and was tender. There was 2 plus bilateral pedal edema.

Chest x-ray on admission showed the heart to be markedly enlarged to the left (Figure 1). Fluoroscopic examination at this time confirmed the presence of enlarged heart but cardiac pulsations were felt to be normal and there was no evidence of pericardial fluid. By repeat fluoroscopy on the fourth hospital day, the cardiac silhouette was markedly enlarged in all directions. The cardiac excursion was diminished and a positive Holmes' sign was observed to be present. The radiologic interpretation was that pericardial fluid was present.

Urinalysis was normal. Complete blood count including the total leukocyte count was within normal limits. The sedimentation rate was 36 mm per hour; total eosinophiles were 175 per cubic mm. Sickle cell preparation, heterophile agglutination titer, antistreptolysin "O" titer, C-reactive protein, cephalin flocculation, total protein

* Fourth year medical student, George Washington University School of Medicine.

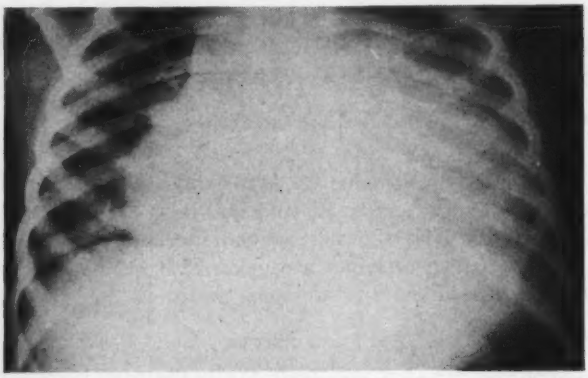

FIG. 1. Markedly enlarged cardiac silhouette on admission

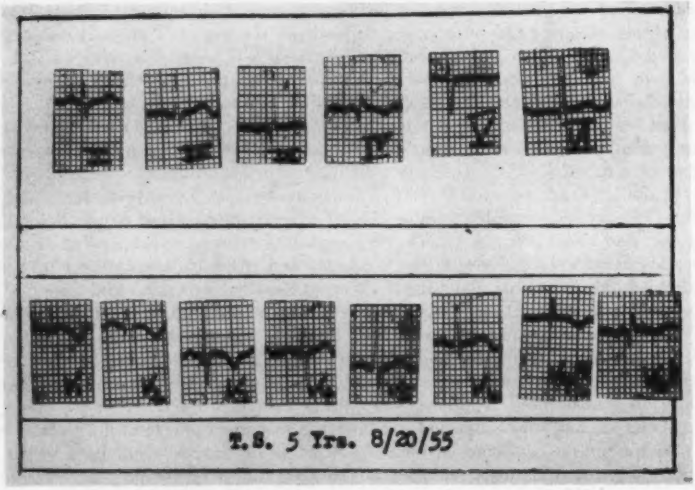

FIG. 2. Electrocardiogram on admission: slight right ventricular hypertrophy as evidenced by a qR in V_1.

and A/G ratio, blood urea nitrogen and spinal fluid examination were all normal. Three blood cultures were sterile. Bone marrow study revealed active hematopoiesis but no other abnormality. Intermediate strength tuberculin test (PPD) performed on admission was negative. Electrocardiogram done on admission revealed slight right ventricular hypertrophy, (Figure 2). The venous pressure on admission was 250 mm. of water.

Treatment of the congestive failure was immediately instituted following admission. The patient was placed in an oxygen tent, digitalized, given thiomerin sub-

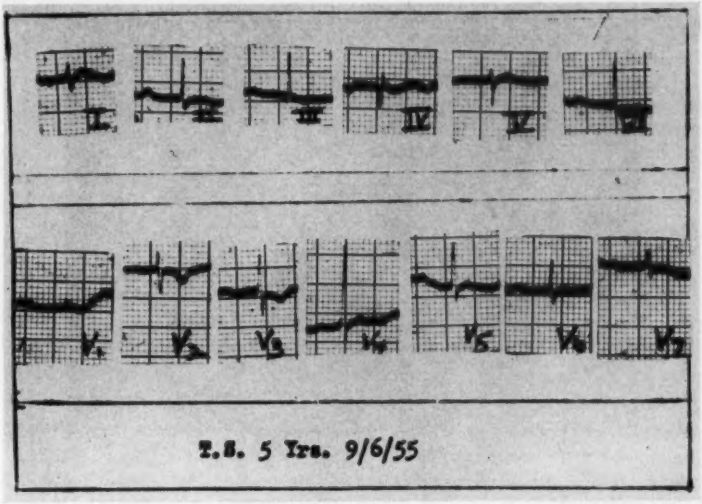

FIG. 3. Electrocardiogram 2 weeks after admission. A cardiac friction rub was present at this time. Generally low flat T waves indicate abnormal myocardium and pericardium.

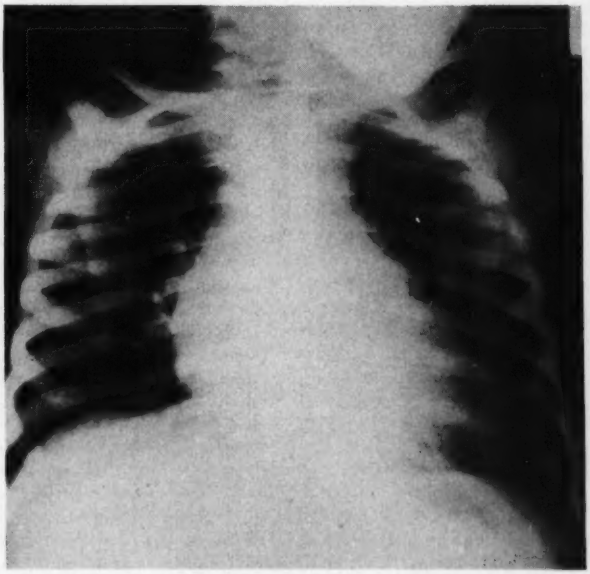

FIG. 4. Marked decrease in size of cardiac silhouette following prednisone therapy and on the day prior to surgery.

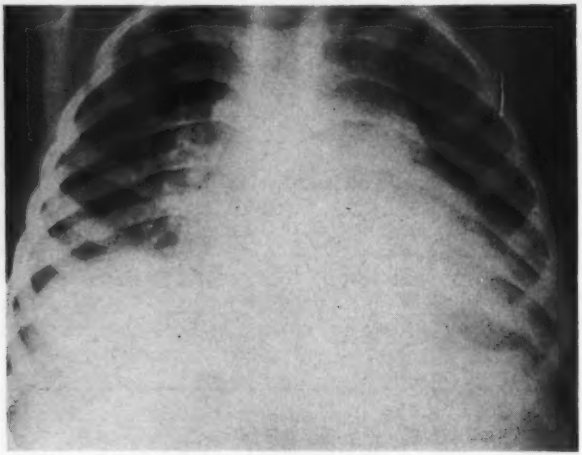

FIG. 5. Cardiac silhouette two and one half weeks following surgery; considerable residual cardiac enlargement.

cutaneously and put on a low salt diet. Response to therapy was dramatic. He lost 9 pounds of weight in the first 24 hours after admission and was clinically much improved. By the third hospital day, his neck veins were no longer distended, respirations were 34 per minute and the liver was no longer palpable. At this time, however, the heart sounds were muffled. This finding, coupled with the abnormal fluoroscopic findings, led to the tentative diagnosis of pericarditis with effusion. In the presence of a negative tuberculin test and with evidence of combined pericardial and myocardial involvement in a child who did not appear toxic enough to have a purulent pericarditis, the most likely etiological possibility was thought to be acute idiopathic pericarditis with effusion. With this impression it was not felt that pericardial paracentesis was necessary particularly since there did not appear to be enough cardiac embarassment from tamponade to warrant it.

The patient was placed on penicillin therapy on admission but continued to have a fever up to 104° daily. Repeat chest x-ray on examination on the fourth hospital day showed that the heart shadow was massively enlarged. Venous pressure at this time was 167 mm of water; with right upper quadrant pressure it rose to 260 mm. Tetracycline therapy was started but the temperature remained elevated (100°–103°). The liver again became palpable and tender. On the seventeenth hospital day a cardiac friction rub was heard. (Fig. 3) At this time prednisone therapy was instituted and the temperature returned dramatically to normal. On the twenty-first hospital day the tuberculin skin test was repeated and found to be definitely positive. The patient was then considered to have tuberculous pericarditis. Accordingly, prednisone was discontinued (with a resultant rise of the temperature to 102°) and therapy with streptomycin and isoniazid was instituted. Pericardial paracentesis was considered but x-ray of the chest showed the heart to have diminished greatly in size. There did not appear to be enough pericardial fluid remaining to justify attempted removal.

On the twenty-sixth hospital day, prednisone therapy was again added to the anti tuberculous therapy, and was continued eight days. On the thirty-eighth hospital

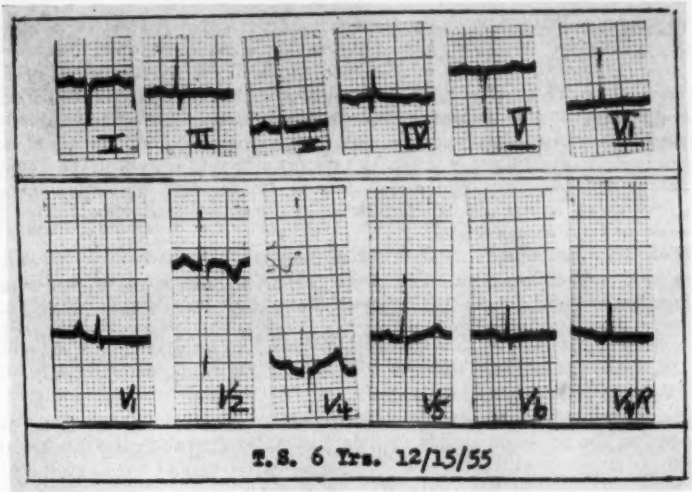

FIG. 6. Electrocardiogram 8 weeks following surgery still demonstrates some evidence of right ventricular hypertrophy in V_1, and continuing evidence of abnormal myocardium and pericardium in the form of generalized low T waves.

FIG. 7. Biopsied specimen. Note the thickness of the pericardium

day it was noted that the patient's abdomen had become enlarged and the neck veins remained distended even in the erect position. The lungs were normal to percussion and auscultation and the heart did not appear to be any larger than it had been for the previous two weeks. (Fig. 4) The liver remained palpably enlarged and abdominal fluid was thought to be present. Fluoroscopic examination performed at this time revealed diminished pulsation of the right heart border with no evidence of fluid. Venous pressure was 230 mm of water. In a further attempt to establish the diagnosis of constrictive pericarditis by demonstrating the typical elevation of the end-diastolic pressure in the right ventricle, cardiac catheterization was attempted but due to venospasm was not successful.

On the fifty-eighth hospital day a pericardiectomy was performed by Dr. Edgar Davis and a thick fibrous pericardium covering most of the heart surface and ranging in thickness from 0.4–1.0 cm was removed. Microscopic examination of this tissue revealed a generalized granulomatous reaction consistent histologically with tuberculous infection. Acid-fast stains revealed acid-fast bacilli morphologically resembling *Mycobacterium tuberculosis*. Culture of this tissue for tuberculous organisms and guinea pig inoculation were negative.

The patient did remarkably well following operation. All signs of cardiac failure disappeared and the heart sounds were normal. Anti-tuberculous therapy was continued. At examination eight weeks after operation the patient was in good health. The heart, however, remained enlarged. (Fig. 5 and 6)

Dr. Sol Katz:

I was impressed that this patient's complaints, in addition to those of pulmonary insufficiency, were abdominal complaints. I think that in pericardial effusion in children we more often see abdominal complaints than we see complaints that indicate the patient has congestive heart failure. These children are often admitted with the diagnosis of "acute abdomen" or acute hepatitis. The astute resident notices that there is venous distension and that is the first clue. I think that most of the children that we have seen with pericardial effusion or constrictive pericarditis have had abdominal complaints as the presenting manifestation or at least prominent among the presenting manifestations. It is also striking that in spite of such a huge heart no murmurs were heard and I am glad that a diagnosis of idiopathic myocarditis was preferred over rheumatic myocarditis, because I think that in a patient who has such a huge heart, whether due to pericarditis or myocarditis, there ought to be significant murmurs if you are going to call it rheumatic. Stated differently, there should be evidence of some endocardial involvement in rheumatic carditis. There are a few exceptions to this rule but I will bow to the cardiologist on that point.

I was a little distressed by what was stated: that there was no evidence of fluid on the first fluoroscopic examination. I am also impressed by the fact that there is a very minimal pulmonary congestion. At least the lung fields are described as fairly clear. I think the diagnosis of acute congestive heart failure would not have been tenable unless one were saying that this

patient has had left ventricular failure but has such marked right ventricular failure that the pulmonary congestion has now been relieved. That would be a tenable explanation. The response to mercurials is not pathognomonic of heart failure but rather indicates that there has been fluid retention that is now responding to mercurials. If you have a patient with cirrhosis of the liver (with an enlarged liver, ascites, evidence of venous obstruction and pedal edema) who is given mercurials, there frequently is a good response and he will lose some of the edema.

The difficulty in doing venous pressure is that one has to understand its limitations and fallacies before one can give an accurate interpretation. The reason you select a zero mark is that theoretically you like that zero mark to be at the level of the right auricle. You can see the difficulty of locating the exact position of the right auricle in a patient such as this. Also this youngster was not lying flat (because of his considerable dyspnea) but was in a semi-reclining position. When the venous pressure determination was repeated there was no assurance that the two determinations were measured in exactly the same way. The most important source of error to me is that there has been no good study of venous pressure measurements in children. The reason for this is that it is hard to say what the level of the right auricle is. We took a number of children and after placing them in the position used for doing venous pressure, x-rayed them in the right lateral view, often with a barium swallow, trying to see where the level of the right auricle was. The variation was tremendous, more so than in the adult. In the adult I would suggest ten centimeters from the back and in children, seven centimeters from the back. You should have a vertical pole alongside the bed, put a piece of tape on it and mark your seven centimeters or whatever else you select as your standard, and say, "as long as this boy is in the hospital, this is going to be his zero mark on this pole". You do not bother measuring from the bed because sometimes you have a sagging mattress; you use your zero mark and keep it for the duration of the venous pressure studies. That is especially true of children.

I will sum up this diversion by saying that even though in truth this was a falling venous pressure it is entirely compatible with cardiac compression due to constrictive pericarditis or pericardial effusion and it is not pathognomonic of heart failure in the sense of there being a left ventricular failure with pulmonary congestion.

A right lateral x-ray view may be very helpful in pericardial effusion. As you know, usually in the lateral film you can see where the inferior vena cava pierces the diaphragm to enter the right auricle. Usually they join at an acute angle. The most dependent part of the pericardial sac is this posterior inferior recess. This is the first place pericardial fluid will accumulate. Very commonly in early pericardial effusion just 50 or 100 cc

will do it and you will find that this angle is obliterated and the angle is convex rather than concave. We always make it a point to do a lateral film on patients in whom we suspect pericardial effusion, especially if it is a small amount.

Tuberculin skin testing was negative. There are several points about that. It may in truth have been a negative tuberculin test. In other words, this may have been a patient who had never been exposed to tubercle bacillus and therefore never developed tuberculin sensitivity. Or it may have been a patient who is anergic, in that because of acute illness or any other thing that can cause anergy, this was a false negative. The third possibility, sometimes seen in first infection tuberculosis, is that the tuberculin test converts from negative to positive from the second to the eighth week of the disease and it may have been in that period when allergy has not fully developed during which the test was done. It is always a good idea, as was done here, to repeat the tuberculin test after a few weeks have elapsed or the patient seems a little better. You may be rewarded by a positive reaction.

Another point I wish to make is that in fluoroscoping the patient in the lateral decubitus position as was done here, sometimes the pericardial sac is really distended and tense, and you do not get nearly as much shift of the pericardial fluid as when the fluid begins to absorb. You then have a succulent sac and there is room for the fluid to stretch and distend.

I think all of us agree that this boy had pericardial effusion and that he had a positive tuberculin test. Two and two mean that he has tuberculous pericarditis but sometimes two and two do not quite add up to four although as a presumption, that is good. Most of the time in tuberculous pleural effusion or idiopathic pleural effusion and tuberculous pericarditis, the diagnosis remains a presumptive one. In the case of pleural effusion we adopt the medical dogma that pleural effusion is due to tuberculosis until proven otherwise. I do not know that anybody could have said that this was not idiopathic pericarditis, presumably an infrequent diagnosis in a child.

I think all pericardial effusions should be tapped. You do not know; this child might have had an influenzal pericarditis, pneumococcal pericarditis or staphylococcal pericarditis at the time he was admitted. I think that getting clear fluid at least makes you feel at that moment that you do not have a purulent pericarditis on your hands. You probably have tuberculous pericarditis but in any event you have some cultures cooking. As infrequently as tuberculous organisms are cultured from pericardial fluid obtained from a patient with tuberculous pericarditis (seldom more than 25 per cent positive cultures on pleural or pericardial fluid), bone marrow culture is even less likely to be positive; gastric washings still less; and

blood culture, of course, least likely to be positive. I do not mean that all these should not be done because if this is tuberculous he has had a parenchymal lesion somewhere in his lungs. Maybe his disease is hematogenous because we do know that there is a hematogenous stage in practically every case of tuberculosis and so it is a good idea to do the bone marrow. It was also a good idea to do the gastric washings but I think the likelihood of these being positive is much less than the likelihood of the pericardial fluid being positive.

If after the injection of air, the heart is perfectly normal in size and on fluoroscopy you can detect rather marked thickening of the parietal pericardium, you have very strong evidence that the pericarditis is tuberculous. In the idiopathic pericarditis that we have seen, even though the heart appears to be of normal size, we have found a perfectly thin, graceful-looking pericardium and the biopsies that we have done have revealed just that; perfectly normal pericardium. In rheumatic pericarditis, you can expect the heart to be enlarged because of the accompanying myocarditis.

At this point, you have two courses open. We have been doing pericardial biopsies on these children and on adults in an effort to help us with the diagnosis. The pericardial biopsy has been a very useful and we think a very innocuous procedure if the patient is not overwhelmingly ill. It can be done expeditiously and is even less hazardous than a pleural biopsy. The other course, and I have no criticism since I think it is the one most everybody uses, is to presume it is tuberculosis and start therapy. The patient often gets well and if he does not, new lesions will develop in the form of lymphadenitis, meningitis or miliary tuberculosis and the diagnosis thus will be made.

The problem of steroids in tuberculosis has been discussed ever since we have had cortisone and if you will look at the brochures that you see on the packages of cortisone you will see a very strong warning not to use these materials in patients who are thought to have tuberculosis. All of you here (and the pediatricians are responsible for this) recognize the value of steroids in patients who have acute, severe, overwhelming, non-tuberculous infections. In severe meningitis I think you routinely use steroids. Well, why not in tuberculosis? The difference is, of course, that at the same time you administer the steroids in these severe non-tuberculous infections you are also using powerful anti-bacterial substances. We now have available powerful anti-bacterial substances as far as the tubercle bacillus is concerned and it would seem entirely logical to use cortisone when the indication is strong. We were brave enough, maybe foolish enough, about two and one-half years ago to begin using steroids at first in patients who were overwhelmingly ill with tuberculosis. We felt that they were going to die and we would not be subject to criticism if we were to use controversial

agents like the steroids. These are patients, who by all criteria, you would consider pre-terminal. We were quite surprised to see that within 24 to 72 hours, their temperature became normal and they were sitting up in bed asking for food. It looked as if they were doing very well. This indicated to us that cortisone had a very powerful anti-toxic activity and as long as the patient was receiving adequate antibacterial therapy, this was good treatment. We then began to expand the use of cortisone, using it in patients who were not so terminal but who were very ill. After all it does little good to give a patient antibacterials if he is going to die before they get to him. Usually when you give streptomycin and isoniazid there is a hiatus between the day of administration and the day the patient responds. Sometimes it will be one to two weeks before this response occurs and if he is going to die before the response occurs you have gained nothing by giving him good anti-bacterial agents. Steroid anti-inflammatory action is quick and often within 24 hours a patient shows a response that you can attribute to the steroids rather than the streptomycin-isoniazid (INH) combination because these antibacterials just do not work that quickly.

The acute exudative manifestations of tuberculosis are usually related to antigen:antibody activity and if there is ever an indication for steroids it would be in such a situation where the response to the allergic component is great and responsible for symptoms. So it would seem logical to use cortisone for the exudative manifestations of tuberculosis (pleural, pericardial, peritoneal or even pulmonary which has a very marked allergic component). I do not advocate their use routinely but I think consideration should be given to steroid use when the patient is very ill. The response of the pleural effusion may be very gratifying, sometimes disappearing within two weeks; that is faster than we ever see it under anti-bacterial therapy.

Will the steroids have an effect in reducing the complication of constrictive pericarditis following anti-tuberculous therapy? Steroids have certainly been demonstrated to inhibit fibroblastic activity with resultant inhibition of fibrous tissue formation. In tuberculous meningitis with block, the English, especially, have demonstrated that intraspinal block can be relieved by cortisone because of this antifibrous tissue response. Theoretically there is a possibility that the use of cortisone in any of these serous membrane diseases will limit the formation of a constrictive envelope.

How long should steroids be continued? The answer must be a little vague. Our indications for steroid therapy have been acute illness of the patient and my own thought is that steroid therapy should be continued until your end point is reached. If the end point is its antitoxic effect, steroid therapy may be discontinued in a matter of a few weeks. If the indication is preventing the formation of a constricting scar, therapy should be continued for a longer period of time. If the indication is overcoming

the anti-allergic response, therapy should be discontinued when the fluid begins to disappear.

As for antibacterial therapy, here you have a difference of opinion as to what should be used. Should it be streptomycin and INH, INH and para amino-salicylic acid (PAS), streptomycin and PAS, or streptomycin, INH and PAS? You have a choice of any of these combinations. The battle still rages as to which of these combinations is the best. Almost everyone agrees that there is no advantage in giving all three drugs over any of the other combinations that I mentioned. I think that there is little question that in a child who is going to be treated with antituberculous therapy INH must be included since you have to be concerned with the possibility of hematogenous phenomena appearing, and as you know, tuberculous meningitis just has not occurred in anybody who has received INH therapy and who did not have meningitis at the start of therapy. I think INH must be one of the drugs. Some people advocate in this situation that you use INH alone. Most people advocate that you use it in combination with PAS or with streptomycin. There is evidence that a combination of INH and PAS is just as good as the combination of INH and streptomycin and it has the advantage of oral adminisration. Most children tolerate PAS very well; they tolerate it better than adults in many of whom gastro-intestinal difficulties develop. There are some theoretical advantages to using INH and PAS or streptomycin and PAS and leaving the third drug as a reserve drug in case the patient suffers a relapse sometime in the future. This is a theoretical rather than a practical suggestion, however.

With intermittent streptomycin therapy and daily PAS our results were just as good as when we used daily streptomycin. Since streptomycin is such a powerful antituberculous drug, I have no criticism of those who want to treat tuberculosis with daily streptomycin therapy for the first two weeks and then when the patient begins to respond, reduce the number of injections to twice weekly. The life.cycle of the tuberculous organism indicates that organism division occurs about every third day so it is just as logical to administer streptomycin therapy every third day. In this child I should think half a gram twice weekly is adequate.

We administer INH in a daily dose of 5 mg per kilogram, in two doses. The blood level falls very rapidly so that after 6 or 8 hours there is practically no measurable amount of isoniazid in the blood. Again, as in the case of streptomycin, blood level of antibacterial is not important; perhaps the tissue level is the crucial factor.

What is the natural history of untreated tuberculous pericarditis? The pericardial effusion may be the sole demonstrable manifestation of tuberculosis or one of several manifestations. The pathogenesis is usually that of tuberculous lymphadenitis (mediastinal) with extension to the pericardium.

Occasionally there is direct extension from the lung parenchyma to the pericardium and more uncommonly a hematogenous spread to the pericardium. There are two groups of patients: one in which the pericardial effusion *seems* to be the only manifestation of the tuberculosis (but as you know from its pathogenesis, another focus is present); a second group with tuberculous pericarditis together with obvious other lesions such as tuberculous pleuritis or miliary tuberculosis, or hematogenous tuberculosis. The usual course is for cardiac compression to develop and cause death. This is often a sequential thing: the patient never quite recovers from the pericardial effusion but goes on to get severe cardiac compression, disseminated tuberculosis and dies. This often occurs in a matter of a few weeks. Actually 85 per cent die, 50 per cent in the first year. There are some patients with tuberculous pericardial effusion who seem to get well spontaneously for a period and then usually about one to one and a half years later acute hematogenous tuberculosis, tuberculous meningitis or tuberculosis in some other location usually kills them.

Other patients develop chronic constrictive pericarditis with or without calcification and eventual chronic cardiac compression occurs. With drug therapy we can expect that most patients will get well. Figures vary but the mortality is 10 per cent or lower in the reported series. The incidence of constrictive pericarditis also varies. Some report 5 per cent. The last report had 30 cases; in 2 of the 30 cases constrictive pericarditis developed.

I think we are seeing a great deal more constrictive pericarditis now than before. Blalock thought there would be less constrictive pericarditis now that we have the antituberculous drugs; actually the opposite is true since many more patients who formerly would have died are now living and getting chronic constrictive pericarditis.

In discussing therapy I think one has to mention the fact that some people advocate surgery in the acute phase. Some recommend that if signs of cardiac compression develop in tuberculous pericarditis, surgery should be done during the acute phase under drug therapy coverage. It seems a little illogical to us because of the low incidence of development of chronic constrictive pericarditis in treated cases of tuberculous pericarditis. If under drug therapy for one to three months, cardiac compression is occurring, then the patient can have decortication. Those who advocate early surgery say that if you wait for cardiac compression and allow it to go on for a long time, the cleavage plane will have been obliterated and degeneration of the myocardial fibers will have occurred, both resulting in a poor outcome of the attempted decortication. This, of course, does not happen overnight, and if the patient is observed up to three months and the cardiac compression is noted to be getting less and less but nonetheless, high venous pressure and an enlarged liver remain, decortication can then be performed with usually very good results.

BORIC ACID POISONING

Aaron Searle, M.D.

In spite of repeated exhortations regarding both the relative uselessness and toxicity of boric acid, its widespread use, both in hospitals, and in the home continues. Another case of boric acid intoxication is reported.

CASE REPORT

J. R., an 18 day old white male infant was admitted to Children's Hospital on September 20, 1955 at 10:00 a.m., 28 hours following his initial ingestion of a formula later discovered to contain powdered boric acid in place of Dextri-Maltose (R). The formula consisted of 8 ounces of evaporated milk, 12 ounces of boiled water, and two tablespoons (about 12 grams) of boric acid powder. He ingested about 2 ounces of the formula at 6:00 a.m., September 19, and immediately after feeding, vomited about one ounce of formula. The net formula retained, therefore, contained about 0.5 gm. of boric acid powder.

In the 24 hours following ingestion, the patient vomited six times, had three loose bowel movements, became apathetic, and fortunately, would not eat.

The remaining history was non-contributory.

Physical examination: When first seen, the patient was active, afebrile, and moderately irritable. He was 6 pounds ½ ounce in weight. The skin was slightly dry, and showed a generalized erythema, most marked on the soles and buttocks. The anterior fontanelle was full, but not bulging. The heart rate was 190–200 per minute. The respirations were rapid and regular for some minutes. It would change to a slower rate for several minutes, then it would revert to its previous rapid rate. The rest of the physical examination was considered normal.

On September 20, the peripheral blood hemoglobin was 11.2 grams per cent, and the hematocrit reading was 41 per cent. The leukocyte count was 27,600 with 36 per cent segmented, and 3 per cent band polymorphonuclear cells; the lymphocytes were 59 per cent. Three days later the peripheral blood hemoglobin was 13.4 per cent, and the hematocrit reading was 46 per cent. The leukocyte count was 5,300, with 30 per cent segmented polymorphonuclear cells; the lymphocytes were 64 per cent.

On September 20, the serum chlorides were 102 meq. per liter, the carbon dioxide combining power being 31 volumes per cent. The urine gave a positive result when tested for boric acid by the flame test but was otherwise normal.

Since the patient could not retain oral feedings, and in order to promote diuresis, intravenous therapy was instituted. Sixty cc. of 1/6 molar lactate was given to ameliorate the acidosis, followed by 100 cc. of plasma, and 50 cc. of saline because it has been shown that patients exhibit a greater tolerance to boric acid poisoning in the presence of plasma and saline.[1]

An exchange transfusion in the manner suggested by Boggs and Anrode[2], was considered, but was not deemed justified in the present case because of the relatively small amount of boric acid ingestion, and the satisfactory condition of the patient.

During the first hospital day, the patient's general condition appeared little altered, except that he was febrile (101.4) at 6:00 p.m., but four hours later he became afebrile without specific treatment. The pulse rate slowed to 170 per min. The abdomen was slightly distended but remained soft and gave rise to no difficulties. At 11:30 p.m., a greenish stool was noted.

The following day, there was considerable improvement. The erythema was much reduced. The patient responded well. The heart rate was 160 per minute. However,

the patient still had to be maintained on parenteral fluids as vomiting still persisted. Hiccough also occurred, for which the patient's stomach was lavaged without any manifest relief. Hiccough terminated without any further treatment. This symptom was also reported by Boggs and Anrode following the first exchange transfusion.

On the third hospital day, the patient began to retain oral feedings. For several hours the patient held his right elbow in extension, and performed repetitive screwing movements with his right hand. The tone in this extremity was markedly increased, while the rest of the limbs had a normal tone. This activity ceased spontaneously, and the tone of the right upper extremity became normal.

On the fourth hospital day, there was a generalized exfoliation of the skin, the previous erythema now having completely disappeared. The pulse rate was now 140 per minute.

The patient was discharged apparently completely well on the ninth hospital day. Weight was now 6 pounds, 2½ ounces.

DISCUSSION

The lethal dose of boric acid is difficult to determine from the literature. Thus Pfeiffer[3] states that the minimal lethal dose is 30 grams, Sollman[4] is of the opinion that "Death may result from the ingestion of 15 to 30 grams of borax or 2 to 5 grams of boric acid". Goodman and Gilman[5] are of the opinion that "Serious poisoning can result from the ingestion of five grams of boric acid".

Boric acid poisoning is fairly common. Goldbloom and Goldbloom[6] were able to find 109 cases including four of their own. In addition, many cases are undoubtedly undiagnosed and are considered to be cases of gastroenteritis, pneumonia, meningitis, atropine poisoning, acrodynia, or Ritter's disease.

The oral route of entrance, as in the case presented, is the most common, but other routes such as subcutaneous clysis, intravenous infusions, topical applications (for burns, skin lesions), and bladder irrigation occur.

The case presented displayed most of the symptoms typical of less severe intoxication. These affect three main systems:

Gastrointestinal: Vomiting and diarrhea are usually the earliest symptoms, and occurred in 73 per cent of the cases described by Goldbloom and Goldbloom.

Skin: An early generalized erythema, especially marked on buttocks, neck, palms, and soles, and perineal regions is followed in one or two days by an extensive desquamation. A skin rash occurred in 88 per cent of Goldbloom and Goldbloom's under two year group. Watson[7] refers to the erythema as a "boiled lobster" appearance.

Central Nervous System: Convulsions occur, involving especially the facial muscles and the extremities. Severe cases progress to coma, cyanosis and shock. Death is ascribed to depression of the respiratory center and shock associated with cerebral edema and congestion[8].

Less common symptoms are referred to two main systems: Cardiovascular—A weak rapid pulse occurs as in the case reported; Urinary—A few cases show oliguria and rarely anuria.

The age group affected ranges from newborns to adults. In Goldbloom and Goldbloom's group, mortality was 70.2 per cent in patients under one year of age with 55 per cent mortality for the group as a whole.

Since the danger of boric acid has become known, many authors have urgently pleaded for its discontinuance. Brooke[9] points out that boric acid usage persists because of its ready availability in drug stores, its recommendation by some physicians and baby care manuals, combined with an established reputation.

As Pfeiffer, et al., put it, boric acid has outlived its usefulness with the advent of antibiotics. There is no reason for retaining boric acid. On the one hand, it is relatively ineffectual, on the other hand it is a potential poison.

Treatment of boric acid intoxication is purely symptomatic:

1) Remove the patient from the source.

2) The stomach should be lavaged if the boric acid was recently ingested.

3) Correct acidosis with sodium lactate and administer chlorides parenterally to counteract displacement of chlorides by boron in the body fluids[10].

4) Give plasma. It has been shown that Ringer's solution 50 per cent, and plasma 50 per cent, increased the tolerance to boric acid.

5) Treat shock if it occurs.

6) Give antibiotics to prevent bronchopneumonia which occasionally occurs.

7) Administer vitamin K[11]. If gastrointestinal bleeding occurs, liver damage may be a factor.

8) For convulsions, sedation must be administered cautiously for fear of adding to an already depressed central nervous system.

SUMMARY

In a child who ingested 0.5 gm. boric acid in his formula, fever, gastrointestinal signs, generalized erythema, and acidosis occurred and were corrected by physiological treatment. The lethal dosage of boric acid may be from 2 to 30 grams, entering the body by any of several routes. There is no practical use for this potentially poisonous substance in pediatric practice.

REFERENCES

1. Pfeiffer, C. C., Hallman, L. F., and Gersh, J.: Boric Acid Ointment: A Study of Possible Intoxication in the Treatment of Burns, J. A. M. A., **128:** 266, 1945.
2. Boggs, Jr., T. R., and Anrode, H. G.: Boric Acid Poisoning Treated By Exchange Transfusion, Pediatrics, **16:** 109, 1955.

3. Pfeiffer, C. C., Emergency Treatment of Common Poisons, Am. J. Nursing, **55:** 1382, 1955.
4. Sollman, T.: A Manual of Pharmacology, Philadelphia, W. B. Saunders Company, 1942.
5. Goodman, L., and Gilman, A.: The Pharmacological Basis of Therapeutics, New York, Macmillan Company, 1941.
6. Goldbloom, R. B., and Goldbloom, A.: Boric Acid Poisoning, J. Pediat., **43:** 631, 1953.
7. Watson, E. H.: Boric Acid: A Dangerous Drug of Little Value, J. A. M. A., **129:** 332, 1945.
8. Young, E. G., Smith, R. P., and MacIntosh, O. C.: Boric Acid as a Poison: Report of 6 Accidental Deaths in Infants, Canad. M. A. J., **61:** 447, 1949.
9. Brooke, C. E., The Boric Acid Problem, G. P., **7:** 43, June, 1953.
10. De Sanctis, A. G., and Varga, C.: Handbook of Pediatric Medical Emergencies, St. Louis, C. V. Mosby Co., 1951.
11. Brooke, C., and Boggs, F.: Boric Acid Poisoning. Am. J. Dis. Child, **82:** 465, 1951.

ORAL INJURY BY BALL POINT PEN

Lee M. Sackett, D.D.S.
John J. Sullivan, D.D.S.

Oral injuries in children resulting from a fall or from being struck while holding various objects in their mouths are all too frequent occurrences. In the authors' personal experience, injuries to the teeth, tears of the buccal gingiva and cheek have been seen. Recently the ball point pen was responsible for a bizarre injury.

CASE REPORT

D. J., a five and one-half year old colored boy was seen in the emergency room, Children's Hospital on September 4, 1955 at 9:15 p.m., with the tip of a ball point pen projecting from the right side of his mouth. It was learned that about three hours prior to admission the patient was running with the pen in his mouth and fell on the floor forcing the pen back into the soft tissues.

On physical examination the boy was in obvious distress. The pen was seen as previously described. The patient could not close his teeth nor could he obtain maximum opening. On intraoral examination it was seen that the blunt end of the pen with the clip had entered the pterygomandibular space for a distance of three centimeters. There was no bleeding from the wound. The teeth and surrounding structures were intact. On palpation of the subauricular region the end of the pen was found to be protruding one centimeter beyond the distal border of the ramus of the mandible. This area was tender to palpation. Because of the danger of the pen clip engaging the soft tissues, no attempt was made to remove the pen at this time.

Examination of posterior-anterior and lateral head roentgenograms made upon admission located the pen clip in the region of the upper right hypopharynx and protruding from the mouth. (Fig. 1).

FIG. 1. Lateral head roentgenogram depicting the depth of penetration of the pen clip.

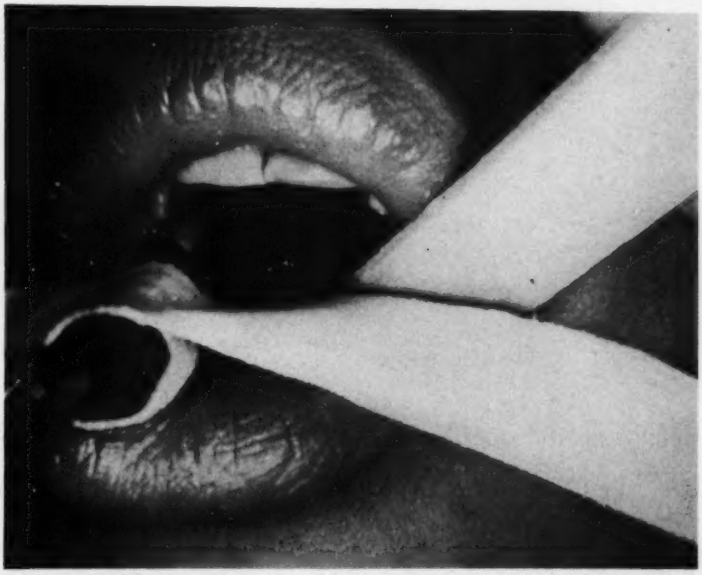

FIG. 2. Photograph showing the point end of the pen protruding from the right side of the mouth and the emergency taping of the pen.

The rest of the physical examination and the laboratory findings were normal.

After examination, the patient was given atropine sulfate, gr. 1/400 and was scheduled for immediate surgery. Meanwhile, in order that the pen's position not be altered it was taped to the face. (Fig. 2). At 2:00 a.m., on September 5, the patient was taken to surgery where intravenous surital sodium and nitrous oxide endotracheal anesthesia were administered. The face, mouth and pen were washed with surgical soap, water and aqueous benzalkonium chloride. Holding the pen in a sterile gauze pad, the wound was enlarged in a superior-inferior direction. The handle of a Bard Parker knife was used to examine the buried portion of the pen in order to discern the position of the pen clip. The clip was located on the lateral aspect of the pen. The wound was then spread in a medio-lateral direction with a curved hemostat and the pen was removed. A small tag of tissue caught under the pen clip was dissected free. The wound was examined for foreign matter and debrided. Two 3-0 catgut sutures were used to close the wound and a one inch piece of rubber dam drain was inserted.

The postoperative course was uneventful. The medication consisted of 0.5 c.c. of tetanus toxoid on admission and procaine penicillin, 600,000 units and warm saline mouthwashes administered twice a day throughout the hospital stay. There was a mild degree of trismus resulting from the spasticity of the internal pterygoid muscle. A slight elevation of body temperature was noted following surgery, (100.2°) but this returned to normal within twenty-four hours. The rubber dam drain was removed on the second postoperative day and the patient was discharged on the fourth postoperative day.

DISCUSSION

Oral injuries due to foreign bodies respond favorably to immediate care consisting of removal of the object, careful debridement and prophylactic antibiotic therapy.

It is interesting to note that in this accident there was direct irritation to the internal pterygoid muscle resulting in spasticity causing both trismus and a locking of the pen in the pterygomandibular space. Probably the one single procedure that prevented serious complications was the immediate taping of the pen in position preventing unnecessary movement and further tearing of the adjacent soft tissues.

Dilute formulas
in newborn period

Today, mothers and babies go home from the hospital much earlier than they did a few years ago. Consequently, formula and feeding instructions for the newborn period assume greater importance.

"The first formula feedings should be relatively dilute," as pointed out by Lee in a recent text,* "and the earliest consideration should be aimed at supplying adequate fluid." Overfeeding in the newborn period "is to be avoided since it frequently leads to digestive disturbances, colic and dietary diarrhea." When the formula is made safely dilute, "most infants can be allowed to take as much of it as they desire to satisfy their hunger."

Lee recommends the feeding of dilute formulas for the first 10 to 20 days of life.

*Lee, H. F., in Lull & Kimbrough: Clinical Obstetrics, Philadelphia, Lippincott, 1953, pp. 630-632.

Dilute feedings for the newborn period are conveniently prepared with Liquid Lactum.

For a formula supplying 10 calories per fluid ounce: use 1 part Liquid Lactum with 3 parts water.

For a formula supplying 13 calories per fluid ounce: use 1 part Liquid Lactum with 2 parts water.

For a formula supplying 16 calories per fluid ounce: use 1 part Liquid Lactum with 1½ parts water.

•

For babies allergic to milk . . .

LIQUID SOBEE® provides a good-tasting *soya formula* that is exceptionally well taken and well tolerated, relieves both gastrointestinal disturbances and eczema, and promotes sturdy growth.

the Mead formula
for general use
in infant feeding

LIQUID **Lactum**

ready-prepared milk and Dextri-Maltose formula

When you prescribe Liquid Lactum, you provide all the nutritional safeguards of a milk and carbohydrate formula in well balanced proportions —plus the convenience appreciated by modern mothers.

Lactum® is the only infant formula that offers all these advantages: authoritative formulation • generous protein • spaced carbohydrate absorption • whole milk nutrients • optimal digestibility • maximum convenience

Let Mead products and services help you in the varied phases of infant feeding

MEAD | SYMBOL OF SERVICE IN MEDICINE

MEAD JOHNSON & COMPANY, EVANSVILLE 21, INDIANA, U.S.A.

MEAD JOHNSON & COMPANY, Suite 419, Eig Building, 8641 Colesville Road, Silver Spring, Maryland. JUniper 9-1222